

visual loss (1%) and 1 patient (0.5%) had hyperperfusion syndrome. The 2 (2%) non-stroke related deaths were from cardio-pulmonary events; the sole (0.5%) neurological death was due to intracranial hemorrhage.

Patients/Arteries	N = 185/201
Minor Stroke	5 (2.4%)
Major Stroke	0
Neurological Death	1 (0.5%)
Non-Stroke Related Death	2 (1%)
Retinal Embolus	2 (1%)
Hyperperfusion Syndrome	1 (0.5%)
All Strokes and Deaths	8 (4%)
All Embolic Related Events	7 (3.4%)

Conclusion: 1) Carotid stenting using neuroprotection devices is feasible. 2) The procedural related embolic complications was 3.4% (minor strokes + retinal emboli) 3) There were no major strokes and 1 fatal intracranial hemorrhage.

Noon

812-5

Angiographic and Pathologic Studies of Late Vascular Responses to Successful Carotid Artery Stenting

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Background: There was little angiographic follow-up data and pathologic studies for patients receiving stents at extracranial carotid arteries. The purpose of this study was to assess the long-term efficacy of carotid artery stenting (CAS) angiographically and histopathologically.

Methods: Out of 29 patients with 36 lesions undergoing 6 months follow-up angiography, 1-2 years follow-up angiography was available in 14 patients with 19 lesions. Quantitative angiographic analysis was performed. Minimal luminal diameter (MLD), interpolated reference diameter and % diameter stenosis were measured. Stent sites were divided into 10 segments. At each segment, minimal diameter and mean diameter were measured. Histopathologic examination was performed in patients undergoing antemortem CAS. Temporal changes in histologic pattern between 8 months and 3 years were examined.

Results: At stenotic segments greater than 50% before CAS, neointimal hyperplasia became greater than other segments at 6 months. Follow-up angiography of 19 lesions at 6 months and 1-2 years revealed a decrease in MLD from 4.41 ± 0.55 mm immediately after stent implantation to 3.45 ± 0.39 mm at 6 months, but no further decrease in diameter after 6 months. Late improvement in luminal diameter was observed after 6 months. (3.45 ± 0.39 mm at 6 months and 3.78 ± 0.3 mm at 1-2 years; $P=0.015$). Micrographs of the stented segment of the 8 months after CAS revealed significant proliferation of smooth muscle cells (SMCs) and abundant deposition of extracellular matrix substance chiefly composed of proteoglycans on the intimal side. In contrast, after 3 years, neointima was primarily composed of dense fibrous collagenous tissue and atrophic SMCs. Lipid core in the atheroma was covered with stable neointima.

Conclusion: Angiographic outcomes up to 2 years after CAS were favorable. Late improvement of neointimal hyperplasia appears to occur after 6 months. Temporal changes in histologic pattern provide a pathologic background for the long-term efficacy of CAS.

POSTER SESSION

1102 Percutaneous Coronary Intervention and Inflammation

Monday, March 18, 2002, Noon-2:00 p.m.

Georgia World Congress Center, Hall G

Presentation Hour: Noon-1:00 p.m.

1102-1

Previous Cytomegalovirus Infection and the Risk of Restenosis After a Strategy of Provisional Stenting

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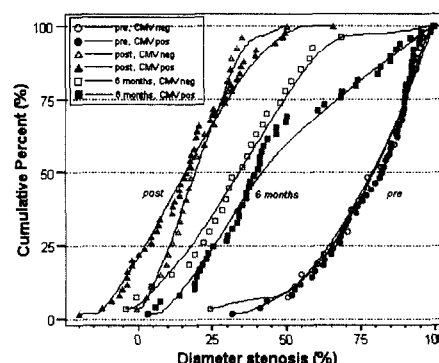
Background: Previous studies have shown that prior infection with cytomegalovirus (CMV) is a predictor of restenosis after atherectomy.

Methods: We prospectively studied 78 consecutive patients scheduled for 6-months follow-up coronary angiography as part of the SIPS study. Anti-CMV IgG and IgM antibodies were measured to determine whether previous exposure to CMV increased the risk of restenosis after a strategy of provisional stenting. In 76 patients (99%), the coronary angiograms before, directly after and 6-months after the intervention could be analyzed quantitatively.

Results: Anti-CMV IgG positive and anti-CMV IgG negative patients had similar minimal lumen diameter (MLD) in the target vessel before and directly after the intervention. After six months, however, the MLD was significantly smaller in CMV-positive as compared to CMV-negative patients (1.57 ± 0.82 mm versus 2.00 ± 0.83 mm, $p<0.03$). Net lumen gain at 6 months was significantly lower in CMV-positive patients (0.89 ± 0.79 mm versus 1.30 ± 0.87 mm, $p<0.04$), and the rate of clinically significant restenosis was significantly higher (31% versus 7%, $p<0.02$). In a multivariate logistic regression model, CMV-seropositivity was an independent predictor of restenosis (odds ratio 5.7 (95%CI 1.2-30.3,

$p=0.04$)).

Conclusion: CMV-seropositivity is an independent predictor of restenosis following coronary intervention.



1102-2

Role of Chlamydia Pneumoniae for Restenosis After Percutaneous Coronary Intervention: The SWICA (Swiss Cardiovascular Center Chlamydia) Trial

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Background: Recently, a reduction of restenosis rate after stenting has been reported in patients with high Chlamydia pneumoniae titers treated with the macrolide antibiotic roxithromycin. The purpose of the present study (SWICA Trial) was to evaluate the effect of clarithromycin for prevention of coronary restenosis in patients with routine percutaneous coronary intervention (PTCA with and without stenting) and concomitant assessment of antibody titers.

Methods and Results: In a randomized, double-blind, placebo-controlled single center pilot study 86 patients with coronary artery disease undergoing PTCA were randomized to receive either standard therapy (placebo group, n=42) or treatment with 2x250 mg clarithromycin for six weeks (treatment group, n=44). Primary endpoint was angiographic restenosis and secondary endpoint major adverse cardiac events (MACE). The patients underwent follow-up angiography after 3 months. Stent implantation was performed in 67% of all cases. Antibody titers were taken at randomization and at the end of the follow-up period. Age, gender, body mass index, history of myocardial infarction as well as cardiovascular risk factors were evenly distributed in the two groups. Diameter stenosis was similar at follow-up (33% vs. 34%, ns) in both groups. Restenosis rate (>50% diameter stenosis) was 23% in the treatment and 21% (ns) in the placebo group, respectively. MACE occurred in 18% and 29% (ns), respectively. Antibody titers for Chlamydia pneumoniae, Cytomegalovirus and Helicobacter pylori were similar in the two groups at randomization and at follow-up without any correlation to restenosis rate. Only Helicobacter pylori titers decreased significantly after antibiotic therapy.

Conclusions: Restenosis rate after PTCA is not influenced by clarithromycin treatment. Seropositivity for Chlamydia pneumoniae, Cytomegalovirus and Helicobacter pylori was not associated with an increased risk of restenosis after percutaneous coronary interventions. Thus, a major pathogenic role of Chlamydia pneumoniae for restenosis after percutaneous coronary intervention appears unlikely.

1102-21

Preprocedural Levels of Acute Phase Reactants Predict an Aggressive Clinical Pattern of Restenosis Following Coronary Angioplasty

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Background: In a subset of patients undergoing coronary angioplasty (PTCA), restenosis is not a benign clinical event but it is associated with recurrence of severe unstable angina (UA), myocardial infarction (MI) or cardiac death (CD). Acute phase reactants are powerful predictors of angiographic restenosis following PTCA, but it is unclear whether they can also identify patients with acute coronary events at long term follow-up.

Methods: We investigated whether pre-procedural plasma levels of C-reactive protein (CRP) and serum amyloid-A protein (SAA) might predict the clinical severity of restenosis at one year follow-up in a group of 111 consecutive patients undergoing successful single-lesion PTCA.

Results: At 1 year, clinical restenosis, defined as recurrence of symptoms and/or positive exercise test and confirmed by the angiographic evidence of > 50% stenosis at the site of PTCA, occurred in 51 patients (46%). Among them, 28 patients (25%) had a more aggressive pattern of restenosis requiring urgent hospital admission because of severe UA (23 patients, 21%) or MI (5 patients, 5%). Recurrence of UA or MI was 46% and 50% in patients with elevated levels of CRP (> 3 mg/L) and SAA (> 5 mg/L), and 2% ($P<0.001$) and 6% ($P<0.001$) respectively in patients with normal levels. Among several clinical, angiographic and procedural variables analyzed by Cox proportional hazards regression model, CRP was the most powerful independent predictor of an aggressive pattern of restenosis (RR=14.3, CI=1.8-111.7, $P=0.011$).

Conclusions: Our data demonstrate that elevated pre-procedural levels of acute phase reactants predict a more aggressive clinical pattern of restenosis, requiring emergent hospitalization. Close post-PTCA surveillance is necessary in these patients.